Chemical burns causing systemic toxicity

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Chemical burns can cause severe local pain and tissue damage which require specialized treatment. They account for one patient in six admitted to a burns unit after an industrial accident (Herbert & Lawrence, 1989). Strong acids and alkalis are the commonest cause. The initial treatment of nearly all such burns is to drench the affected area with water, remove contaminated clothing and continue drenching. There is however a theoretical danger of explosion if water is used to remove certain alkali metals (sodium, potassium, rubidium and caesium) because they liberate hydrogen gas and heat. Strong acids and alkalis also evolve heat when water is added, but the heat is dissipated rapidly if sufficient water is used.

The local effects of chemical tissue destruction can be very severe and may distract attention from the dangers of systemic toxicity, which are greatest for burns due to yellow (white) phosphorus, hydrofluoric acid and phenols.

Yellow phosphorus is an important intermediate in the production of phosphorus pentoxide, phosphoric acid and phosphorus chloride and sulphide. It is also used in tracer bullets. It ignites in air to produce the acid gas phosphorus pentoxide. Wounds should therefore be drenched and then left immersed in water or covered with wet dressings. The phosphorus particles can be removed with forceps and stored under water. They are easier to see if washed or swabbed with a 1% copper sulphate solution, when black copper sulphide is formed, followed by 1% sodium bicarbonate solution to neutralize any phosphoric acid. Only dilute copper sulphate solution should be used, to avoid systemic absorption of copper ions, which may result in haemolysis (Summerlin et al., 1967).

In 1966 in Vietnam there were sporadic reports of sudden deaths within 24 h of injury in soldiers burned by white phosphorus over as little as 10% of the body surface area (Bellamy, 1988). Bowen et al. (1971) subsequently showed that animals which died following experimentally-induced phosphorus burns usually did so in the first 18 h and had higher serum phosphorus and lower serum calcium levels than survivors. Phosphorus poisoning causes hypocalcaemia, partly through a disturbance of renal tubular function (Cushman & Alexander, 1966). Close moni-

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toring of serum calcium and phosphate levels as well as the electrocardiogram is needed during the first 48 h (Kaufman *et al.*, 1988). Intravenous calcium may be required to prevent arrhythmias. Absorption of phosphorus can be reduced by early effective decontamination. This can be facilitated by placing water filled baths at all sites where phosphorus might be spilled (Cooke, personal communication).

Hydrogen fluoride is highly corrosive and in one study was the second most common chemical agent causing burns that required admission to a burns unit (Herbert & Lawrence, 1989). It is widely used in the glass, metal polishing and semi-conductor industries. The severe local reaction is due to the ability of the fluoride ion to penetrate lipid barriers (Edelman, 1986). The fluoride ion forms insoluble salts with calcium and magnesium; hence hypocalcaemia and hypo-magnesaemia (Burke *et al.*, 1973) can occur if fluoride is absorbed. These effects have been noted as early as 30−45 min after exposure (May & Gross, 1985). Cardiac arrhythmias and death have been reported from hydrofluoric acid burns (Chan Kwok-Ming *et al.*, 1987; Mullett *et al.*, 1987), involving as little as 2.5% of body surface area (Tepperman, 1985) and hypocalcaemia should be considered in any hydrofluoric acid burn involving over 1% body surface area (Greco *et al.*, 1988), particularly if there has been some inhalation of hydrofluoric acid fumes. (Inhalation of fumes alone may cause systemic toxicity if the solution contains more than 60% hydroflouric acid [Greco *et al.*, 1988].) There is intense local pain, thought to be caused by nerve stimulation from the resultant excess of potassium ions at the nerve endings (Klauder *et al.*, 1955).

The amount of hydrofluoric acid absorbed will be reduced by prompt and effective flooding of the contaminated skin with water and by subsequent application of topical calcium gluconate gel to chelate the fluoride. Topical gel will not affect any fluoride absorbed into the subcutaneous tissues and local infiltration with calcium gluconate solution is required (Iverson *et al.*, 1971). Intra-arterial injection and regional intra-venous instillation may also help (Velvart, 1965).

Phenol (carbolic acid, lysol) was once widely used as an antiseptic and commonly taken as a suicidal agent (Reid *et al.*, 1907). It is powerfully corrosive and is absorbed rapidly through the skin and gastro-intestinal tract. The lethal dose is lower after skin absorption than after ingestion (Goldfrank *et al.*, 1990). Death has been reported within 10 min of sustaining a 25% body surface area phenol burn (Griffiths, 1973). Methaemoglobinaemia, renal failure (Chan, 1971) and laryngeal oedema (Klein & Little, 1983) have all been recorded following cutaneous absorption, and metabolic acidosis can occur. Phenol is used in plastic surgery practise as a chemical peeling agent and ventricular dysrhythmias have been noted during such procedures (Warner & Harper, 1985). In one series, 10 of 43 patients suffered some dysrhythmia (Trappman & Ellenby, 1979). Any solution of phenol of 2% or more is potentially dangerous (Goldfrank *et al.*, 1990). Although systemic toxicity appears to be related to area and duration of exposure, concentration is the main determinant of local skin damage. Phenol provokes coagulative necrosis which may slow its penetration through the epidermis. Dilution reduces the necrosis and therefore leads to increased absorption (Connary & Hayes, 1970). The concurrent destruction of nerve endings makes some burns painless resulting in prolonged exposure. Physical removal is invariably ineffective. It is recommended that phenol

burns are swabbed with large volumes of polyethylene glycol 300 (70 parts PEG, 30 parts industrial methylated spirits) for a minimum of 10 min. Polyethylene glycol penetrates the epidermis and dissolves the retained phenol (Connary & Hayes, 1970). If polyethylene glycol is not available immediately, then deluge washing with water should begin at once. A detergent added to the deluge will help remove the phenol. After phenol exposure, urine output, urinalysis and renal function should be monitored.

In all cases of chemical burns the examining doctor should consider the systemic effects of cutaneous absorption of the burning agent as well as the risks of ingestion and inhalation. Rapid, effective decontamination, followed by careful monitoring and treatment with specific agents are the most effective means to minimise the risk of systemic toxicity.

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